units could be correlated with the incidence of this disease. As units became stationary and their level of sanitation improved, the incidence of the disease decreased. This conclusion seems to be indicated by the observation that in the fall of 1945 infectious hepatitis was not epidemic in American Forces in Italy, despite the fact that most of the troops were newcomers to the Theatre. By that time sanitation was being maintained at a high level. It is believed that one of the reasons why this disease was epidemic in the forward units of the 5th Army in the fall of 1944 was the impossibility, under fighting conditions, of maintaining any real sanitary measures. One cannot escape this conclusion from a study of the natural history of infectious hepatitis in our forces in North Africa and Italy.

The aim of the Medical Corps in respect to the treatment of this disease was to get the men back to duty in condition so that they would continue on duty, in as short a time as possible. It was found that bed rest, coupled with a high protein, low fat diet containing fresh vegetables which was acceptable to the patients, brought about the desired result. It might be stated at this point that lean beef and dried milk powder were found to be excellent and acceptable sources of protein. Another important factor in returning these patients to duty was a period of physical rehabilitation. Patients convalescent from the disease and with essentially normal liver function as determined by the bromsulfalein test, were given graded exercises over a period of ten days ending up with a day of fairly heavy exercise. If they went through this period without clinical or laboratory evidence of recurrence or relapse of the disease, then we felt quite sure that they would be able to go back to any type of duty which might be required of them. By putting our convalescent patients through this course of exercise we were able to pick up those who would have suffered a relapse after they had returned to their units. This system of therapy and rehabilitation permitted the return to full duty of over 90 per cent of the patients who had infectious hepatitis.

## *Abstracts of Further Discussion*

Dr. Sheila Sherlock (Postgraduate Medical School of London, England) reported on aspiration liver biopsy study over the last five years of nine patients with cirrhosis developing after infective hepatitis. In the acute phase disorganization of the reticulin frame-work of the hepatic lobule is essential for consequent cirrhosis.

In three patients the liver lesion has remained well compensated. In two patients, seen soon after the acute illness, parenchymal damage was present. One of these has died of liver failure. In four patients hepatic fibrosis is prominent with associated portal hypertension. Two of these have died of gastrointestinal hemorrhage.

Infective hepatitis seems an important etiological factor in classical Laennec's cirrhosis.

Dr. S. Karelitz (Mount Sinai Hospital). In March and April of 1947 we experienced an unusual outbreak of acute fulminating hepatitis resulting in death through acute yellow atrophy of the liver in four babies and active hepatitis in two more. The first case was a 2½-year-old Puerto Rican female who had been burned on February 13. Thirty-one days later she became ill with an unexplained fever and an enlarged liver and spleen. She developed jaundice and within a few days died in cholemia.

On April 3, 1947, a 2½-month-old boy (RW) was readmitted to the hospital because of jaundice and fever of one day duration. His liver was large, the icteric index was 63, the serum alkaline phosphatase was 84 King-Armstrong units, and the urine contained bile. The child lived for

five days. Post mortem examination revealed acute yellow atrophy of the liver. He had been in the hospital because of alimentary intoxication beginning February 8, 1947, and received plasma transfusions on February 10 and on February 14 or 47 to 51 days prior to the onset of jaundice.

Another child, 2 months of age (AT) was admitted on April 3, 1947, because of jaundice and a petechial eruption over the entire body. This child died 8 hours after admission. He too had been in the hospital in February because of alimentary intoxication and received 60 cc. of plasma on February 22, February 24, and March 11, thus 37 days intervened between the first plasma transfusion and the onset of jaundice.

On April 4th (N. M.), a 6-week-old female was admitted because of a febrile illness and pallor for the previous 3 or 4 days. She had received 595 cc. of Rh negative blood during an exsanguination transfusion for erythroblastosis fetalis on February 22 at Mount Sinai Hospital. Her icteric index mounted from 12 on admission to 99. She too had bile in the urine (4 plus). Her serum alkaline phosphatase was 36 King-Armstrong units and the serum bilirubin 7.8 mgm. per cent. She died on the 8th day after admission. Her post mortem examination revealed the identical pattern of acute liver necrosis as observed in the other three patients. All four had positive cephalin flocculation reactions.

Because of the rarity of acute yellow atrophy in this age group, the occurrence of the disease within a very short period in babies who had received plasma on the pediatric wards of Mount Sinai Hospital we felt that the source of the infection was the plasma except in the baby who had had the replacement transfusion.

All children who had been on the pediatric ward at the time these children were there were investigated and several were seen at the follow-up clinic. Of the 20 who received plasma half of them were brought back. One of these had had a febrile illness with jaundice and recovered. Another baby previously treated for alimentary toxicosis developed a very large liver and spleen and had several periods of unexplained fever since her discharge from the hospital. She recovered spontaneously. Cephalin flocculation and Thymol turbidity tests were done on the blood of the babies seen at the follow-up clinic. The results were negative in all except the two who had 2 plus cephalin flocculation reactions.

This outbreak of serum jaundice is significant because of the high mortality, the high incidence of the disease in such young babies, and the relatively short incubation period as compared to that usually observed. A detailed clinical report of these observations will be published with Drs. M. H. Bass and Ralph Moloshok and the pathological findings will be reported by Dr. P. Klemperer.